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Docket No. 47653.1 (1789)

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

Applicant:	J.C. Houck, et al.		
Serial Number:	09/189,130	Art Unit:	1631
Filed:	November 10, 1998	Examiner:	M. Borin
For:	SMALL PEPTIDES AND METHODS FOR TREATMENT OF ASTHMA AND INFLAMMATION		

CERTIFICATE OF MAILING

I hereby certify that this paper (along with any paper referred to as being attached or enclosed) is being deposited with the United States Postal Service as first class mail in an envelope addressed to: Commissioner for Patents, Washington, D.C. 20231 on December 7, 2000.

Barbaranne Jenness

Barbaranne Jenness

Honorable Commissioner for Patents
Washington, DC 20231

Sir:

DECLARATION OF JOHN LIPANI, M.D.

I, John Lipani, hereby declare that:

1. I am a citizen of the United States of America residing at and presently I am .Chairman of the Scientific Advisory Committee of Histatek, LLC, the assignee of the present application.

2. I hold a M.D. degree from Tulane University. I held a Fellowship in Rheumatology at the University of Washington and had a clinical practice as a rheumatologist for about 18 years, during which I conducted clinical research in

connection with a variety of new anti-inflammatory compounds. Prior to my present position at Histatek, I was Vice President, Clinical Development for Abgenix, Inc. andGroup Director of Inflammatory Disease Development for Smith Kline Beecham. Thus, I have been involved in development efforts for numerous compounds in the anti-inflammatory field and have over 27 years experience in research and development in the anti-inflammatory field. A copy of my curriculum vitae is attached hereto as Attachment A.

3. I have read and understand the Office Action of June 28, 2000, including the references cited therein.

4. The present invention is directed to a pharmaceutical composition having anti-inflammatory activity comprising a pharmacological carrier and an anti-inflammatory effective amount of a peptide having the formula f-Met-Leu-Phe-Phe.

5. Based on my knowledge and experience in the field, it is my opinion that, prior to the present invention, it was well known to those skilled in the art that formyl methionyl peptides have pro-inflammatory activity.

6. However, surprisingly, the present inventors have discovered that f-Met-Leu-Phe-Phe, can provide a useful anti-inflammatory effect.

7. In the Office Action dated June 28, 2000, the examiner states:

> . . . the essential difference [is] the effect of a biological mediator (such as F-Met peptide) when it is used alone as compared to its use in the presence of another pro-inflammatory agent. Cellular response to f-Met peptides (which can be described as inflammatory response) is the same type of reaction which mediates response of the organism to a foreign infection. It is well known in the art that biological mediators such as chemotactic factors stimulate the migration of neutrophils from circulation into sites of infection or tissue damage. These mediators are also believed to increase cell adhesion to injured sites and to activate neutrophils to release toxic agents such as oxygen metabolites and proteases. Thus, in the presence of a provoked infection the response caused by f-Met peptides have protective, anti-inflammatory function.

8. It is true that the prior art teaches that:

> Cellular response to f-Met peptides (which can be described as inflammatory response) is the same type of reaction which mediates response of the organism to a foreign infection. It is well known in the art that biological mediators such as chemotactic factors stimulate the migration of neutrophils from circulation into sites of infection or tissue damage. These mediators are also believed to increase cell adhesion to injured sites and to activate neutrophils to release toxic agents such as oxygen metabolites and proteases.

However, those responses are "pro-inflammatory" responses. The claimed composition of the present invention blocks those responses. Thus, the claimed composition has an "anti-inflammatory" response.

9. The examiner concludes:

> Thus, in the presence of a provoked infection the response caused by f-Met peptides have protective, anti-inflammatory function.

This conclusion is erroneous. First, based on my knowledge and experience in the art, there has never been even a hint of a suggestion that a doctor should treat an infection with fMLP. Indeed, such a treatment would aggrevate the pro-inflammatory

response already caused by the infection and crate further damage to tissue. Further, characterizing this effect as anti-inflammatory is completely erroneous.

10. The examiner states further that:

> An example of an agent which, similarly to f-met peptides, can be pro- or anti-inflammatory was given in the previous Office action: effects of colony-stimulating factor (CSF) are similar to those of formyl peptides. See, e.g., Beaulieu et al., Wright et al.. CSF is one of theleading mediators of inflammation. See, e.g., al-Janadi et al. At the same time CSF is being used to treat inflammation. See, e.g., Burak et al.

Contrary to the examiner's statement, based on my knowledge and experience in the art, CSFs are not used to treat infection. The CSFs are administered to support patients with overwhelming infection who do not have the white cells to mount a defense. The CSFs stimulate specific lineages of white cells in the bone marrow deficient patient. CSFs are not *pre se* inflammatory mediators as is TNF or Il-1, etc. (Nemunaitis J, Blood, 1991)

11. Based on my knowledge and experience in the art, it is my opinion that no doctor would administer a pharmacological composition to induce a "pro-inflammatory" response.

12. The examiner also states and concludes that:

> Characteristically, . . . the effect of the claimed composition is demonstrated only as inhibitor of inflammatory effect caused by another f-Met peptide, fMLP. The absence . . . of showing of the effect of fMLPP alone is not surprising because Kermode shows (Table 2) that fMLPP (the peptide of the claimed composition) is more potent chemotactic agent and stimulator of neutrophil degranulation than fMLP (the peptide used

as "pro-inflammatory" agent). One would expect that fMLPP, alone, would be at least as "pro-inflammatory" as fMLP.

13. However, Kermode conducted *in vitro* tests using rabbit neutrophils that were suspended in solution containing salts, BSA, buffer and for some tests glucose. We have found that such *in vitro* tests are not a predictor of the bioactivity of fMLPP *in vivo*. Although based on the teachings of the prior art, " [o]ne would expect that fMLPP, alone, would be at least as 'pro-inflammatory' as fMLP," as concluded by the examiner, that is an erroneous expectation. Further, based on the teachings of the prior art, one of ordinary skill in the art would not expect fMLPP to act any differently after prior treatment with fMLP.

14. Based on my knowledge and experience in the art, there is not a direct relationship between *in-vitro* observations and *in-vivo* effects. The statements by the examiner based on Kermode ignore the complexities of the *in-vivo* state, such as feedback loops and redundant pathways.

15. HK-X is a small peptide that Applicants discovered to have anti-inflammatory effect, and which has been found to be antagonistic to both the formyl peptide receptor (FPR) and the integrin VLA-6. Although it is related to a family of inflammatory molecules (e.g., f-met-leu-phe), HK-X (f-met-leu-phe-phe) has been shown to block inflammatory pathways utilizing G-protein signaling and the α-6 moiety of VLA-6.

16. It is my understanding that the Examiner contends that, based on Kermode et al, "one would expect that fMLPP, alone, would be at least as "pro-inflammatory" as fMLP." The experiments conducted by Dr. Clagett show the opposite result, that is, fMLPP is anti-inflammatory and also inhibits the pro-inflammatory effects of fMLP.

17. Based on my knowledge and experience in the field, it is unacceptable to use an inflammation promoting agent (e.g., f-met-leu-phe) as a therapeutic agent.

18. I have read the Declaration of Dr. Clagett that is being submitted in response to the outstanding Office Action and I agree with the conclusions and opinions expressed by him.

19. I further declare that all statements made herein of my own knowledge are true and that all statements made on information and belief are believed to be true; and, further, that these statements were made with the knowledge that willful false statements and the like so made are punishable by fine or imprisonment, or both, under Section 1001 of Title 18 of the United States Codes, and that such willful false statements may jeopardize the validity of the application or any patent issued thereon.

11-26-00
Date

John Lipani, M.D.

ATTACHMENT A

CURRICULUM VITAE

NAME: **John A. Lipani, M.D.**

BIRTHPLACE AND DATE: Staten Island, New York
1940

SOCIAL SECURITY NUMBER: 098-38-5978

OFFICE ADDRESS: Histatek LLC
37 St. Germain Street
San Francisco, CA 94114

EDUCATION:

1962	B.A., Villanova University Villanova, Pennsylvania
1962-1963	Graduate Studies in Chemistry New York University New York, New York
	M.D., Tulane Medical School New Orleans, Louisiana
1967-1968	Internship, Virginia Mason Hospital Seattle, Washington
1968-1969	Internal Medicine Residency, The Mason Clinic Seattle, Washington
1969	Aerospace Medicine School Seattle, Washington
1971-1972	Internal Medicine Residency, The Mason Clinic Seattle, Washington
1973	Fellowship in Rheumatology, The Mason Clinic Seattle, Washington
1995	Financing and Accounting, The Wharton School University of Pennsylvania

LICENSURE AND CERTIFICATION:

1967	Louisiana State Medical License
1971	Washington State Medical License
1972	Certified by the American Board of Internal Medicine

PROFESSIONAL PRACTICE AND ADMINISTRATIVE EXPERIENCE:

1969-1971	Chief of Aerospace Medicine

	Chanute Air Force Base, Illinois
1971	Interim Hospital Commander Chief of Aerospace Medicine Da Nang Air Base, Republic of Vietnam
1972-1973	Chief Resident, Medicine The Mason Clinic Seattle, Washington
1972-1994	Clinical Teaching Appointments: Clinical Associate Professor of Medicine University of Washington Seattle, Washington
1974-1989	Private Practice of Rheumatology
1977-1979	Office Director, Internal Medicine Group (A multispecialty practice including Cardiology, Pulmonology, Oncology, and Rheumatology)
1976-1989	Clinical Research for Sandoz, Ives, Adria, Norwich-Eaton, Perdue-Frederick, Warner-Lambert, Berlex, Syntex, Upjohn, Pfizer, Ciba-Giegy, Wyeth-Ayerst, Robins, Beecham, Lederle, Riker, Greenwich Pharmaceutical Companies. (Primary investigator on over 100 protocols studying NSAIDs and antihypertensives)
1977-1985	Medical Advisory Panel Upjohn Company Kalamazoo, Michigan
1978-present	Legal consultant and expert witness. Cases involving rheumatologic problems, personal injury, medical malpractice; Department of Labor and Industries, State of Washington
1979-1982	Director, Arthritis Resource Center Providence Medical Center Seattle, Washington
	Director, Rheumatology Clinical Fellowship Program Providence Medical Center
1979-1985	Rheumatology Consultant Beckloff Associates, Inc. Overland Park, Kansas
1984-1989	President LCS Research Management Edmonds, Washington

(Professional Practice and Administrative Experience Continued)

1989-1991	Associate Director of Clinical Research Immunex Corporation Seattle, Washington
	Responsible for GM-CSF, soluble cytokine receptor clinical trials; successful PLA for GM-CSF
1991-1992	Medical Director, Rheumatology Norwich Eaton Pharmaceuticals, Inc. Norwich, New York
	Responsible for NSAID and Bisphosphonate Clinical Development
1992	Associate Director Rheumatology and Immunology Clinical Research Centocor, Inc. Malvern, Pennsylvania
	Responsible for phase II and III programs using anti-CD4 monoclonal antibodies; phase I/II programs for anti-TNF monoclonal antibody
1992-1993	Director, Inflammation & Tissue Repair Clinical Research Development and Medical Affairs North America, SmithKline Beecham
	Responsible for 5 products; a marketed NSAID (Relafen®) and 4 products, phases I to III.
1993-1997	Group Director, Inflammation and Tissue Repair Clinical Research, Development and Medical Affairs, North America SmithKline Beecham King of Prussia, Pennsylvania
	Responsible for 6 products; one marketed NSAID (Relafen®) in Phase IV and 5 products in phase I to III.
	Responsible for the clinical development of 5 products in phases I and II and one marketed product. Member Worldwide Arthritis Advisory Committee for strategic product development. Other responsibilities include: analysis/review of in-licensing candidate molecules; and, marketing copy approval for the marketed asset. Member, steering

committee for joint venture of anti-CD4 monoclonal antibody development, IDEC and SB Pharmaceutical companies.

(Professional Practice and Administrative Experience Continued)

1997-1999

Vice-President, Clinical Development
Abgenix Inc.
Fremont, CA

Responsible for clinical, manufacturing, assay development, regulatory, data management and project management for all development phase products

1999-Present

Consultant to the Biopharmaceutical Industry. Areas of expertise include: Rheumatology/Immunology clinical development; antibody development; safety monitoring; phase I to III clinical development; anti-inflammatory phase IV planning.

Consulting activities July,1999 to present:

CHIRON, INC., Emeryville, CA. Assignments included Phase II conduct of clinical trials in Osteoarthritis using IGF-I intra-articularly. Pre-clinical development planning for anti-CD40 in multiple inflammation indications.

Contact: Bruce Scharschmit, M.D. 510-923-3830

HISTATEK LLC, San Francisco, CA. Chairman, Scientific Advisory Committee. Pre-clinical development of the lead molecule in multiple indications.

Contact: Craig Palmer, Ph.D. 415-564-1235

2000 - Present

President/COO
Histatek LLC

Responsible for research and development activities including basic science, pre-clinical and clinical development; investor relations; executive management team; and, member of the Board of Directors.

MEMBERSHIPS:

Northwest Rheumatism Society

King County Medical Society

American College of Rheumatology, Founding Fellow

American Lupus Society

American Society of Hematology

Drug Information Association

APPOINTMENTS:

PMA Education and Research Institute. Faculty for Drug Development in Rheumatology 1993, 1994.

Industrial Relations Committee, American College of Rheumatology, 1994.

Advisory committee to OMERACT (Outcomes Measurement in Rheumatology), 1994, 1995, 1996, 1997, 1998, 1999, 2000

Clinical Safety Working Group, OMERACT, 1995 to Present

Program Committee for Innovative Therapies Conference March in 1996, 1997, 1998, 1999, 2000

PUBLICATIONS:

1. The Arthritis of Hypothyroidism. **Lipani JA** and Leonard JL. Submitted for publication and presented at the Northwest Rheumatism Society Meeting, Seattle, Washington, 1975.
2. Preference Study of the Hypnotic Efficacy of Triazolam 0.125 mg. Compared with Placebo in Geriatric Patients with Insomnia. **Lipani JA.** Current Therapeutic Research, 24:4, 8/78.
3. The Use of Fenclofenac in Rheumatoid Arthritis: An Open Label Study. Presented at the Ninth European Rheumatology Congress, Wiesbaden, West Germany, 9/9/79. **Lipani JA.** Published the Royal Society of Medicine International Congress and Symposium Series No. 28, 1980, Page 67.
4. Lupus and You. A Handbook for Patients. **Lipani JA.** American Lupus Society, 1981.
5. Pathogenesis of Rheumatoid Arthritis. **Lipani JA.** Presented at Bolivian-American Medical Society. Seville, Spain, 11/81.
6. Six-Month Multicenter Study Comparing Nabumetone with Naproxen in the Treatment of Osteoarthritis. Pisko J, **Lipani J**, et al. American Journal of Med 83:86, 12/87.
7. Controlled Evaluation of Nabumetone in the Treatment of Active Adult Rheumatoid Arthritis-Nabumetone vs. Naproxen Double-Blind Parallel Study. Vasey FV, **Lipani JA**, et al. American Journal of Med 83:55, 12/87.
8. Rhu GM-CSF Treatment of Neutropenia in Glycogen Storage Disease. Hurst D, **Lipani J**, et al. Submitted for publication American Society of Hematology 32nd

Annual Meeting, Boston, Massachusetts, 11/90.

9. Concurrent use of GM-CSF and Foscarnet after Allogeneic Marrow Transplantation (BMT). Selvaggi K, Przepiorka D, **Lipani J**, et al. Submitted for publication American society of Hematology 32nd Annual Meeting, Boston, Massachusetts, 11/90.
10. Hematologic Improvement in a Patient with Smouldering Leukemia after Treatment with Recombinant Granulocyte-Macrophage Colony Stimulating Factor. Sparano JA, **Lipani JA**, et al. Submitted for publication Annals of Internal Medicine, 1990.
11. Phase I/II Trial of Recombinant Human Granulocyte-Macrophage Colony Stimulating Factor Following Allogeneic Bone Marrow Transplantation. Nemunaitis J, Buckner D, **Lipani J**, et al. Blood, 77:2065 5/91.
12. Monoclonal Antibodies: Technical Development and Therapeutic Indications. In: *Rational Drug Design*, CRC Press, Philadelphia, PA, 1992. Dalesandro M, Sanders M, **Lipani J**. 1995, p. 73.
13. Refractory Osteoarthritis: Differential Diagnosis and Therapy. Mandell BF and **Lipani JA**. Rheum Dis Clinics. 21:163, 2/95.
14. Clinical Update of the Relative Safety of Nabumetone in Long-Term Clinical Trials. Poland M, **Lipani JA**. J. Inflammopharmacology 3:35, 1995.
15. Relationship Between Antibodies Against Human Soluble Complement Receptor 1 (hsCR1) from Various Species. Ruggieri P, Kaplan J, Everitt D, **Lipani J**, Jorkasky D, Boike S, DeClement F, Moore F, Herzyk D. J Clin Immunol 16:97, 1996.
16. The Use of CE9.1, a Primatized Monoclonal Anti-CD4, in the Treatment of Rheumatoid Arthritis. In: *Novel Therapeutics Agents for the Treatment of Autoimmune Diseases*. CRC Press, Philadelphia, PA. 1997. Yocum DE, Solinger, AM and **Lipani JA**. 1997, p. 65.
17. Comparative Effects of Nabumetone, Soludac and Ibuprofen on Renal Function. Cook ME, Wallin JD, Thakur VC, Kadowitz PJ, McNamara BD, Garcia HM, **Lipani JA**, Poland M. J. Rheu. 24:6, 1997.
18. Clinical and Immunologic Effects of a Primatized Anti-CD4 Monocolonal Antibody in Active Rheumatoid Arthritis: Results of a Phase 1, Single Dose, Dose Escalating Trial. Yocum DE, Solinger AM, Tesser J, Gluck O, Cornett M, O'Sullivan F, Nordensson K, Dallaire B, Shen CD and **Lipani JA**. J. Rheumatology 25:1257, 1998.

ABSTRACTS:

1. Perforations, Ulcers and Bleeding (PUBs) in a Large, Randomized, Multicenter Trial of Nabumetone Compared with Diclofenac, Ibuprofen, Naproxen and Piroxicam. Espinoza LR, **Lipani J**, Poland MP. Presented at ILAR, Barcelona, Spain, 1993.
2. Efficacy and Safety of Nabumetone in the Elderly Versus Diclofenac, Piroxicam, Naproxen and Ibuprofen. Morgan GJ, Lebanon NH, Poland MP, **Lipani J**. Presented at ILAR, Barcelona, Spain, 1993.
3. Do all NSAIDs have a Linear Dose Response for Gastrointestinal Side Effects?

Eversmeyer W, Poland MP, **Lipani J**. Presented at ILAR, Barcelona, Spain, 1993.

4. Activities and Lifestyle Index as a Tool to Evaluate NSAIDs in Arthritis. Pincus T, Poland MP, **Lipani J**. Presented at ILAR, Barcelona, Spain, 1993.
5. Safety of NSAIDs: Are all NSAIDs Created Equal? Lister B, Poland MP, **Lipani J**. Presented at ILAR, Barcelona, Spain, 1993.
6. Hemoglobin Determination is a Simple, Cost-Effective Predictor of Symptom-Free PUBs in the NSAID Treated Population. Poland MP, **Lipani J**. Presented at ILAR, Barcelona, Spain, 1993.
7. Cost-effectiveness analysis of preventing perforations, ulcers and bleeds associated with nonsteroidal anti-inflammatory drugs. Bloom BS, **Lipani JA**, Schainbaum S, DeMarinis R. Presented at American College of Rheumatology, Minneapolis, Minnesota, October 1994.
8. Clinical activity in an early phase I trial of primatized® IDEC-CE9.1-an anti-CD4 monoclonal antibody-in RA. Solinger AM, Yocum DE, Tesser J, Gluck O, O'Sullivan F, Henkel C, Cornett M, **Lipani J**. Presented at American College of Rheumatology, Minneapolis, Minnesota, October 1994.
9. Modulation of Mitogen and Recall Antigen Proliferation by Anti-CD4 monoclonal Antibody: Results of a multi-dose study. Yocum DE, Murarescu M, Soundararajan D, Nordensson K, Solinger AM, **Lipani J**. American College of Rheumatology, Poster Session, San Francisco, October 1995.
10. Results of a Multi-Dose Protocol 7002 using and Immunomodulating, Non-depleting Primatized Anti-CD4 Monoclonal Antibody in Rheumatoid Arthritis (RA). Kaine J, Solinger A, Yocum D, **Lipani J**, Klas P, Tesser J, Wiesenhutter C, O'Sullivan F, Shuman S, Rigby W. Presented at American College of Rheumatology, San Francisco, 1995.
11. Clinical Update of the Relafen Safety of Nabumetone in Long-Term Clinical Trials: Proposed Rationale for Safety Profile. Poland M, **Lipani J**. Presented at the Ivth International Meeting on Side-Effects of Anti-Inflammatory Drugs. Sheffield University, UK, August 1995.
12. Treatment of Rheumatoid Arthritis with a Primatized® Anti-CD4 Monoclonal Antibody, SB-210396 (IDEC-CE9.1) - Results of an open-label extension study in patients responding to induction therapy. Tesser J, Wiesenhutter C, Levy R, Schiff M, **Lipani J**, Solinger A, MacDonald B, Elliot M. Arth Rheum 40:S22H, No. 1160, September 1997.
13. ABX-CBL for the treatment of steroid refractory Acute Graft Versus Host Disease (GVHD). Deeg, HJ, Bolwell, B, Long G, Scheuning, F, Rifkin, R, Cunningham, J, Blazer, B, Furlong, T, Havrilla, N, **Lipani J**. Presented at the IBMTR Transplant Symposia, Keystone, CO, March 1999.
14. A Phase II Multi-center Trial of ABX-CBL in Patients with Steroid Resistant Acute Graft Versus Host Disease (GVHD). Rifkin, R, Deeg, HJ, Bolwell, B, Long, G, Scheuning, F, Cunningham, J. Blazar, B, Havrilla, N, **Lipani J**. Presented at ASCO Atlanta, GA, May, 1999.

15. Clinical Trials of a fully human anti-Il8 antibody for the treatment of psoriasis. Lohner M, Krueger G, Gottlieb A, **Lipani J**, et al. Presented at the European Cogress of Dermatology, Dec. 1999.

PATENTS:

Therapeutic Agents against CD147 for Immune Conditions such as Graft Versus Host Disease. Inventors: Lipani, J, Havrilla, N, Davis, CG, Hales, J.

Human Antibodies to Interleukin-8. Inventors: Davis, CG, Lipani, J, Hales, J, Lohner, M.

SELECTED INVITED LECTURES:

1994 Refractory Osteoarthritis, Novel Treatment Modalities. Rheumatology Rounds, Cleveland Clinic, Cleveland, OH, May, 1994.

1995 Anti CD4 Monoclonal Antibodies in the Treatment of Rheumatoid Arthritis, New York Hospital for Special Surgery, New York, NY, March, 1995.

How May We Define "Induction Therapy". Biologic Agents in Autoimmune Disease IV, San Francisco, March, 1995.

Clinical Update of the Relative Safety of Nabumetone in Long-term Clinical Trials: Proposed Rationale for Safety Profile. Sheffield University, UK, August, 1995.

Clinical Aspects of COX-2 Selective NSAIDs: Safety Profile of Nabumetone, NSAID Update, Bergen, Norway, 1995.

Are Monoclonal Antibodies a Viable Treatment for RA? American College of Rheumatology. Biologic Study Group, San Francisco, CA, 1995.

1996 Practical Assessment of Long-Term Toxicity. FDA Rheumatoid Arthritis Workshop. Rockville, MD, March, 1996.

Anti-CD4 Monoclonal Antibodies in the Treatment of Rheumatoid Arthritis. Rheumatology Rounds, The Cleveland Clinic, Cleveland, OH, April, 1996.

1997 Workshop on Designs of Clinical Programs in Rheumatology: Cooperative Effort with FDA and Academia. The Perspective of the Sponsor. Jan 17, 18, 1997, Taipei, Taiwan.

CMR Workshop: Safety Evaluation of Biotechnologically-Derived Pharmaceuticals. The View of the Clinician, February 1997, West Sussex, UK.

1998 Understanding the mystery of pharmaceutical drug development and the FDA. The Cleveland Clinic, Cleveland, OH, January 1998.

-Clinical Research Design Seminar, Rheumatology Fellowship, Cleveland Clinic, January 1998.

Anti-IL8 in the treatment of psoriasis. IBC Conference. Washington, DC, Dec.

1998

1999 Anti-CD147 and a Therapeutic Target. Innovative Therapies Meeting. San Francisco, CA March, 1999.

Thoughts on the preclinical Evaluation of Immunomodulatory Therapies: A clinician's view. Covance sponsored conference on pre-clinical development. Washington, DC. Sept., 1999.

The Rheumatologist in Industry. Mentoring session. American College of Rheumatology, Annual Meeting, Boston, MA. Nov., 1999.